AMENDMENTS

In the claims:

Claims 1-3 (Cancelled)

Claims 4-15 (Canceled).

16. (Previously Presented) A method of inhibiting a binding event between a target protein (T) and a binding protein (P) in a host, comprising:

administering to said host an effective amount of a non-naturally occurring bifunctional inhibitor molecule (I) of less than 5000 daltons consisting essentially of:

(a) a target protein ligand that specifically binds to a target protein (T); and

(b) a blocking protein ligand that specifically binds to a blocking protein (B) ,

wherein said target protein ligand and said blocking protein ligand are covalently bonded to each other, optionally through a linking group;

in order to non-covalently bind the target protein (T) and the blocking protein (B) to produce a tripartite complex (T-I-B) that prevents access of the binding protein (P) to the target protein (T).

17. (Original) The method according to Claim 16, wherein said bifunctional inhibitor molecule comprises a linking group.

18. (Previously Presented) The method according to Claim 16, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein that is also bound by said binding protein (P).

19. (Previously Presented) The method according to Claim 16, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein (T) that is not bound by said binding protein (P).

20. (Original) The method according to Claim 16, wherein said tripartite complex is produced intracellularly.

21. (Original) The method according to Claim 16, wherein said tripartite complex is produced extracellularly.

22. (Previously Presented) The method according to Claim 16, wherein said blocking protein (B) is endogenous to said host.

23. (Previously Presented) The method according to Claim 22, wherein said blocking protein (B) is selected from the group consisting of: peptidyl-prolyl isomerases, Hsp90 (Heat shock protein 90), steroid hormone receptors, cytoskeletal proteins, albumin and vitamin receptors.

24. (Previously Presented) The method according to Claim 16, wherein said bifunctional inhibitor molecule (I) is administered as a pharmaceutical preparation.

Claims 25-39 (Cancelled)

Please enter the following new claims:

40. (New) A method of inhibiting a binding event between a target protein (T) and a binding protein (P) in a host, comprising:

administering to said host an effective amount of a non-naturally occurring bifunctional inhibitor molecule (I) of less than 5000 daltons consisting essentially of:

(a) a target protein ligand that specifically binds to a target protein (T) with a binding affinity of at least about 10^{-4} M; and

(b) a blocking protein ligand that specifically binds to a blocking protein (B) with a binding affinity of at least about 10^{-4} M,

wherein said target protein ligand and said blocking protein ligand are covalently bonded to each other, optionally through a linking group;

in order to non-covalently bind the target protein (T) and the blocking protein (B) to produce a tripartite complex (T-I-B) that prevents access of the binding protein (P) to the target protein (T).

41. (New) The method according to Claim 40, wherein said bifunctional inhibitor molecule comprises a linking group.

42. (New) The method according to Claim 40, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein that is also bound by said binding protein (P).

43. (New) The method according to Claim 40, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein (T) that is not bound by said binding protein (P).

44. (Newl) The method according to Claim 40, wherein said tripartite complex is produced intracellularly.

45. (New) The method according to Claim 40, wherein said tripartite complex is produced extracellularly.

46. (New) The method according to Claim 40, wherein said blocking protein (B) is endogenous to said host.

47. (New) The method according to Claim 46, wherein said blocking protein (B) is selected from the group consisting of: peptidyl-prolyl isomerases, Hsp90 (Heat shock protein 90), steroid hormone receptors, cytoskeletal proteins, albumin and vitamin receptors.

48. (New) The method according to Claim 40, wherein said bifunctional inhibitor molecule (I) is administered as a pharmaceutical preparation.

49. (New) A method of inhibiting a binding event between a target protein (T) and a binding protein (P) in a host, comprising:

administering to said host an effective amount of a non-naturally occurring bifunctional inhibitor molecule (I) of less than 5000 daltons consisting essentially of:

(a) a target protein ligand that specifically binds to a target protein (T) with a binding affinity of at least about 10^{-4} M; and

(b) a blocking protein ligand that specifically binds to a blocking protein (B), wherein said blocking protein ligand is a peptidyl-prolyl isomerase ligand,

wherein said target protein ligand and said blocking protein ligand are covalently bonded to each other, optionally through a linking group;

in order to non-covalently bind the target protein (T) and the blocking protein (B) to produce a tripartite complex (T-I-B) that prevents access of the binding protein (P) to the target protein (T).

50. (New) The method according to Claim 49, wherein said bifunctional inhibitor molecule comprises a linking group.

51. (New) The method according to Claim 49, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein that is also bound by said binding protein (P).

52. (New) The method according to Claim 49, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein (T) that is not bound by said binding protein (P).

53. (Newl) The method according to Claim 49, wherein said tripartite complex is produced intracellularly.

54. (New) The method according to Claim 49, wherein said blocking protein (B) is endogenous to said host.

55. (New) The method according to Claim 49, wherein said bifunctional inhibitor molecule (I) is administered as a pharmaceutical preparation.

56. (New) A method of inhibiting a binding event between a target protein (T) and a binding protein (P) in a host, comprising:

administering to said host an effective amount of a non-naturally occurring bifunctional inhibitor molecule (I) of less than 5000 daltons consisting essentially of:

(a) a target protein ligand that is known to specifically bind to a target protein (T); and

(b) a blocking protein ligand that is known to specifically bind to a blocking protein (B),

wherein said target protein ligand and said blocking protein ligand are covalently bonded to each other, optionally through a linking group;

in order to non-covalently bind the target protein (T) and the blocking protein (B) to produce a tripartite complex (T-I-B) that prevents access of the binding protein (P) to the target protein (T).

57. (New) The method according to Claim 56, wherein said bifunctional inhibitor molecule comprises a linking group.

58. (New) The method according to Claim 56, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein that is also bound by said binding protein (P).

59. (New) The method according to Claim 56, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein (T) that is not bound by said binding protein (P).

60. (Newl) The method according to Claim 56, wherein said tripartite complex is produced intracellularly.

61. (New) The method according to Claim 56, wherein said tripartite complex is produced extracellularly.

62. (New) The method according to Claim 56, wherein said blocking protein (B) is endogenous to said host.

63. (New) The method according to Claim 62, wherein said blocking protein (B) is selected from the group consisting of: peptidyl-prolyl isomerases, Hsp90 (Heat shock protein 90), steroid hormone receptors, cytoskeletal proteins, albumin and vitamin receptors.

64. (New) The method according to Claim 56, wherein said bifunctional inhibitor molecule (I) is administered as a pharmaceutical preparation.